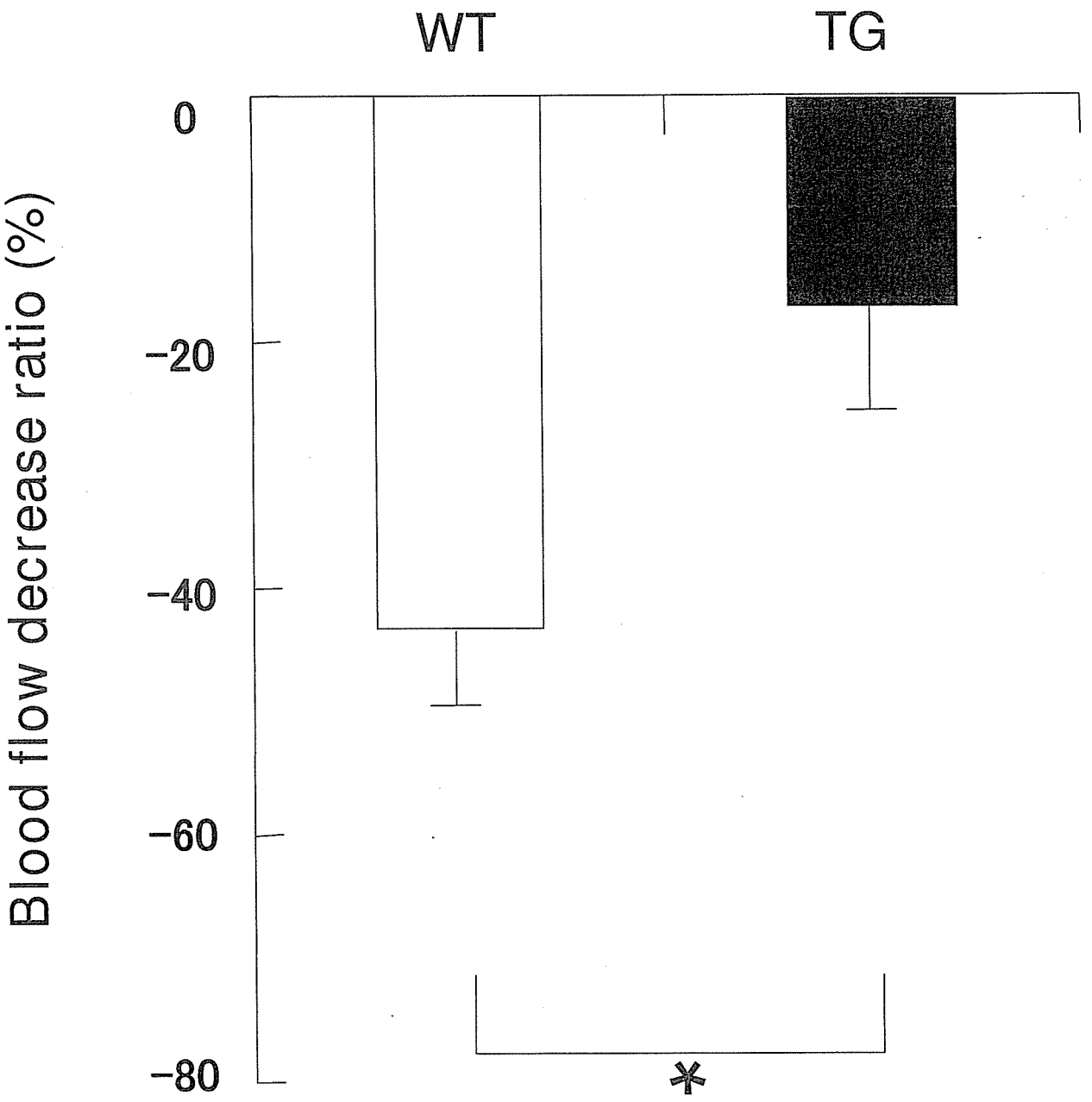
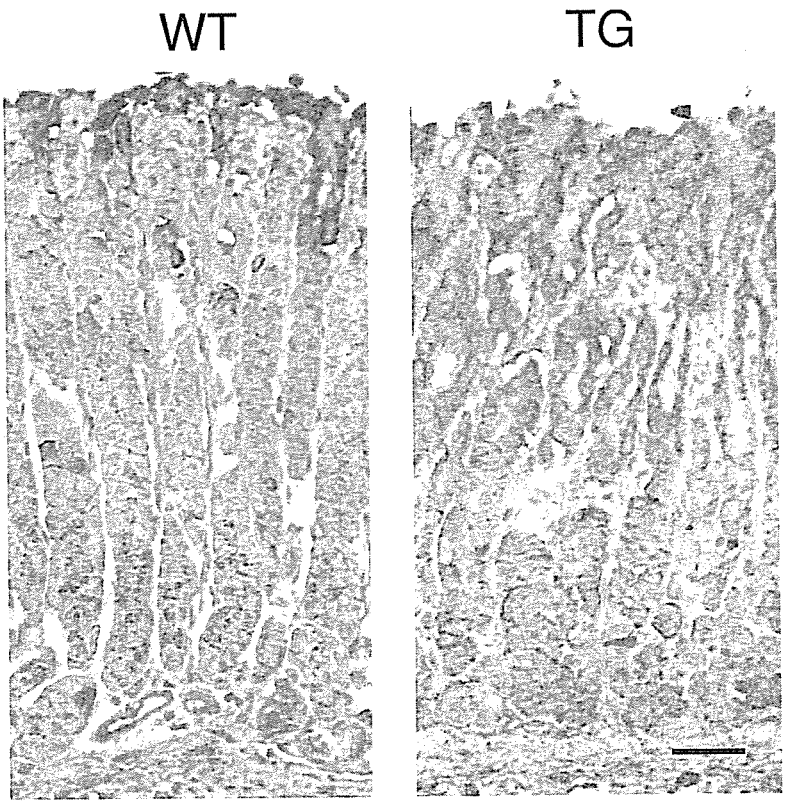
Figure 3



A



B

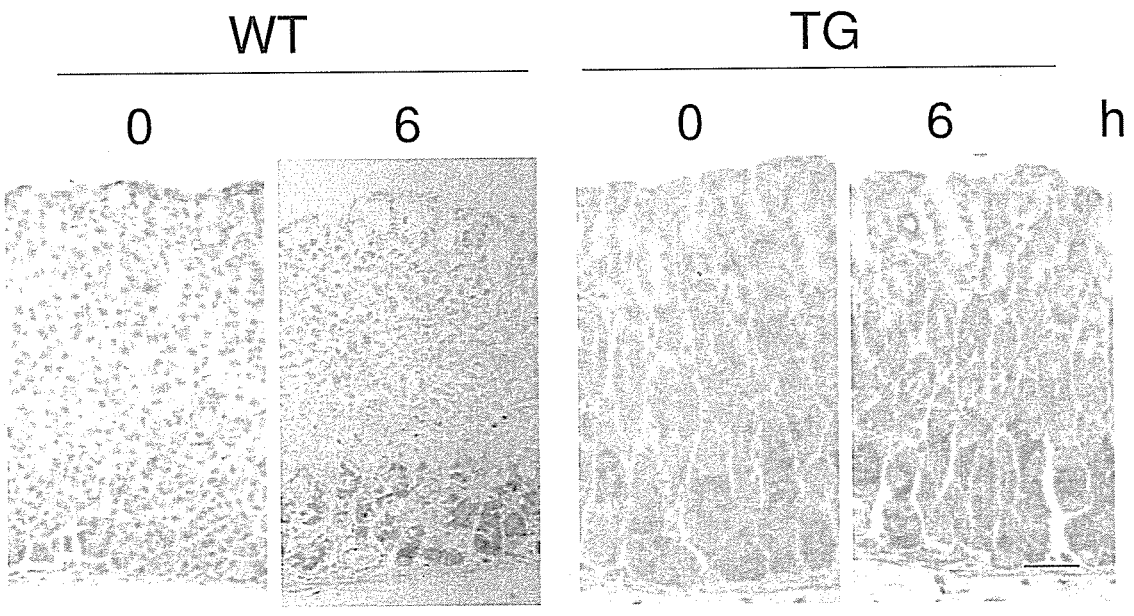
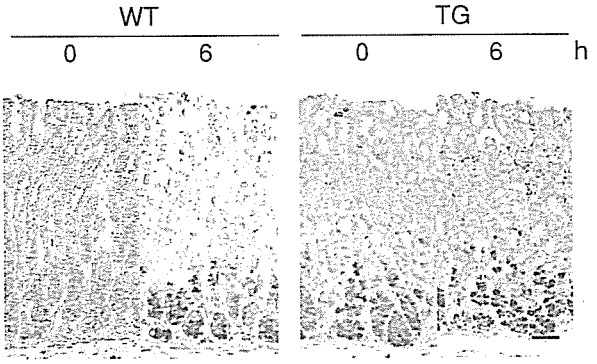
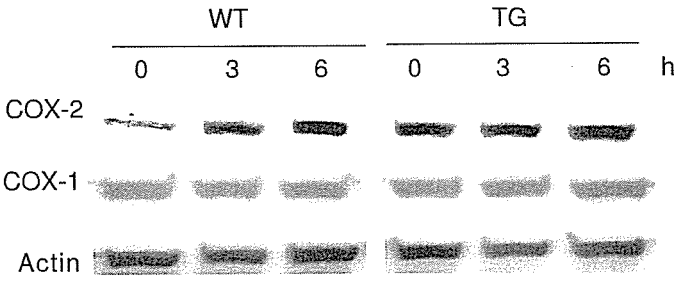


Figure 4

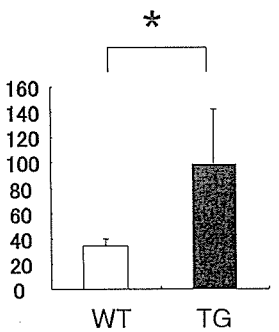
A



B



C



D

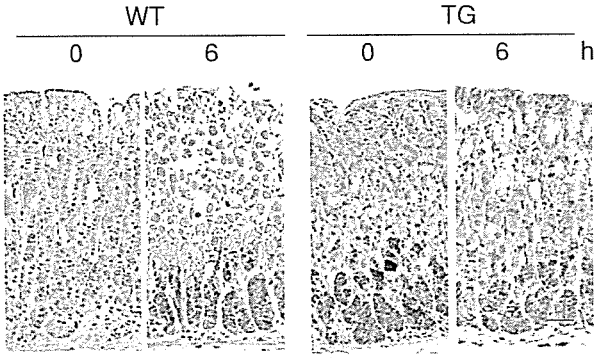
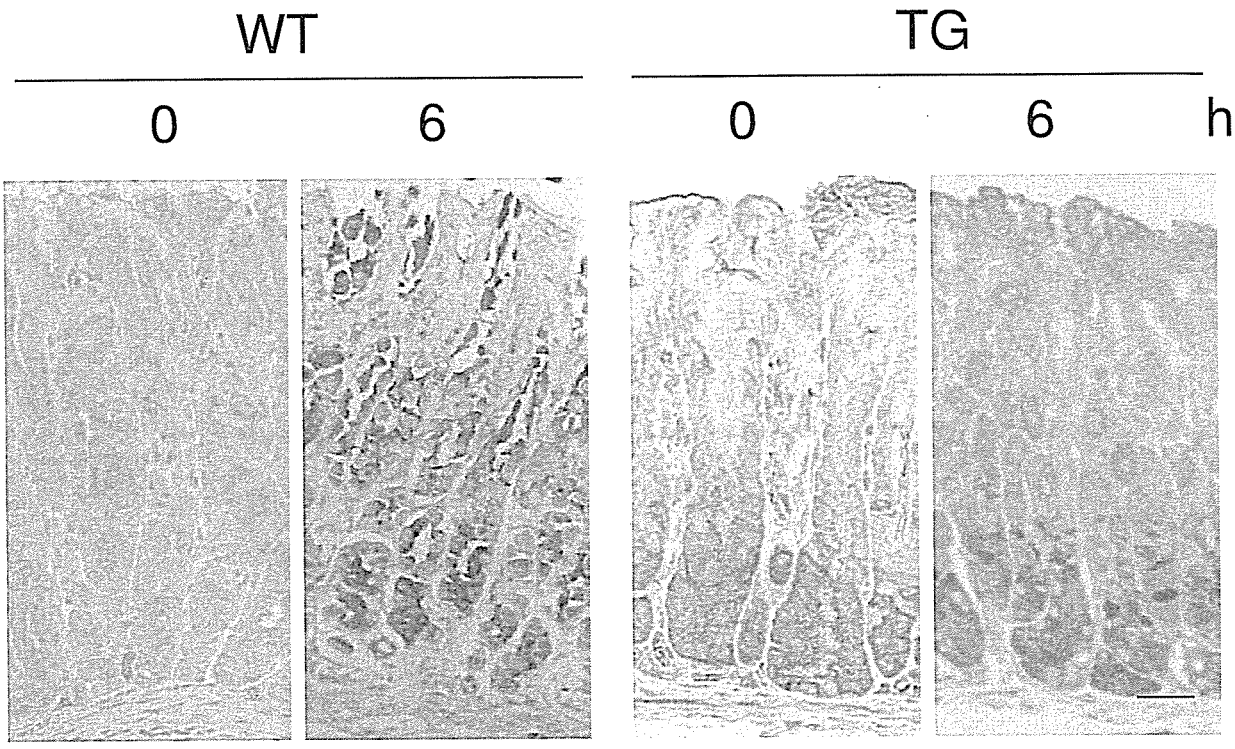


Figure 5

a



b

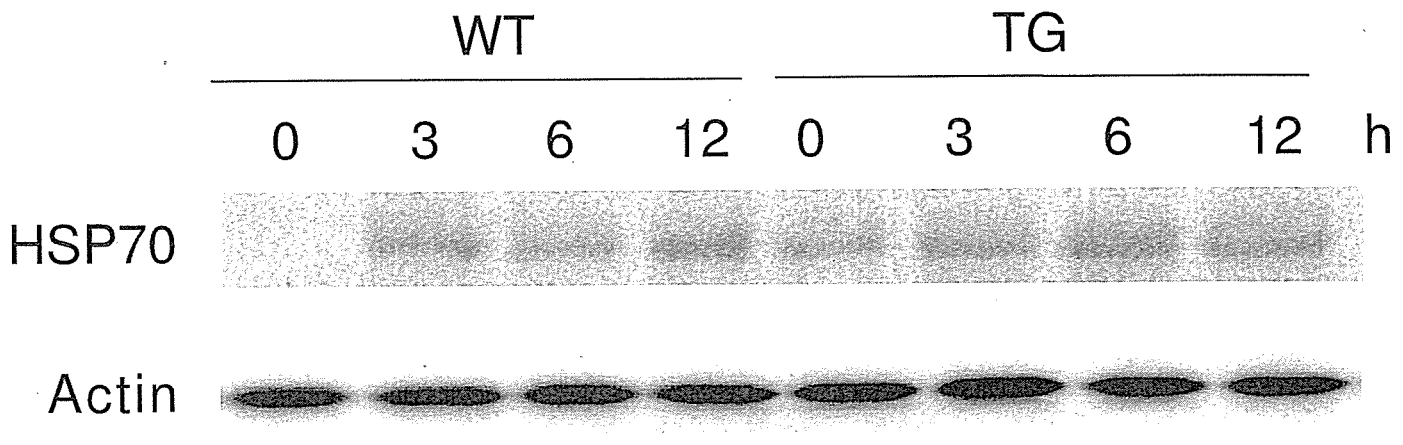


Figure 6

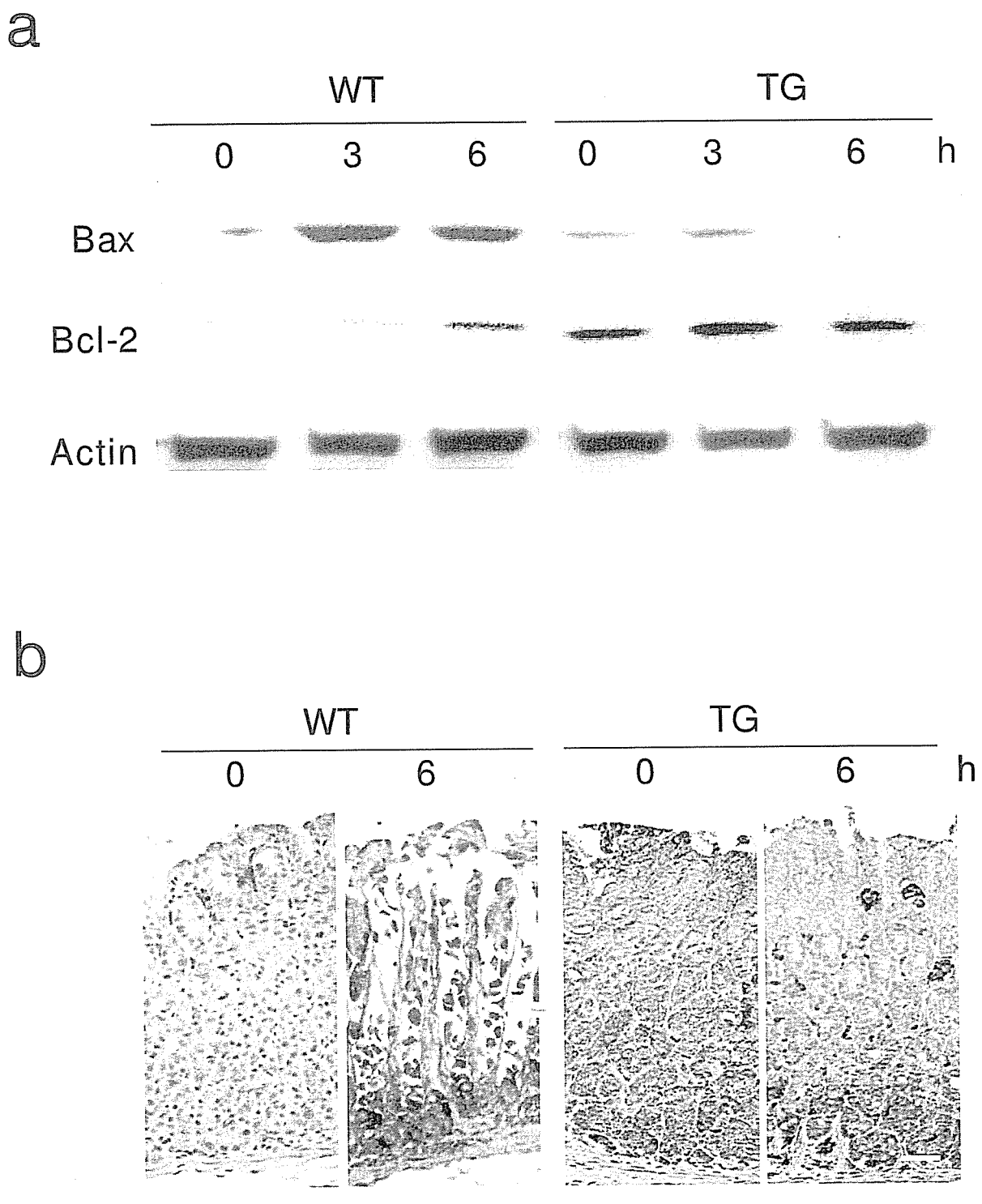

a
WT
TG
0
3
6
0
3
6
h
Bax
Bcl-2
Actin
b
WT
TG
0
6
0
6
h
c
WT
TG
0
6
0
6
h
Procaspase-3
(32 kDa)
Cleaved
Caspase-3
(17 kDa)

Figure 7

7. Brogan PA, Davies R, Gordon I, et al. Renal angiography in children with polyarteritis nodosa. Pediatr Nephrol 2002;17:277-83.
8. Mnif N, Chaker M, Oueslati S, et al. Abdominal polyarteritis nodosa: angiographic features [in French]. J Radiol 2004;85:635-8.
9. Ewald EA, Griffin D, McCune WJ. Correlation of angiographic abnormalities with disease manifestations and disease severity in polyarteritis nodosa. J Rheumatol 1987;14:952-6.
10. Okada M, Konishi F, Sakuma K, et al. Perforation of the sigmoid colon with ischemic change due to polyarteritis nodosa. J Gastroenterol 1999;34:400-4.
11. Arkin A. A clinical and pathological study of periarteritis nodosa. A report of five cases, one histologically healed. Am J Pathol 1930;6:401-26.
12. Tanakaya K, Konaga E, Takeuchi H, et al. Penetrating colon ulcer of polyarteritis nodosa: report of a case. Dis Colon Rectum 2001;44: 1037-9.
13. Streicher JH. Massive gastrointestinal bleeding due to solitary simple gastric erosion (of Dieulafoy). Ger Med Mon 1966;11:448-52.
14. Inaguma D, Kurata K, Ishihara S, et al. A case of MPO-ANCA-related vasculitis that recurred as gastrointestinal bleeding and presented difficulty in treat [in Japanese with English abstract]. Jpn J Nephrol 1998; 40:560-5.

Third Department of Internal Medicine, Fukuoka University School of Medicine, Fukuoka, Japan.

Reprint requests: Kazuhiro Maeda, MD, Third Department of Internal Medicine, Fukuoka University School of Medicine, Nanakuma 7-45-1, Jonan-ku, Fukuoka-city 814-0180, Japan.

0016-5107/$32.00
doi:10.1016/j.gie.2005.09.003

Ruptured duodenal varices after endoscopic ligation of esophageal varices: an autopsy case

Takashi Machida, MD, Ken Sato, MD, PhD, Akira Kojima, MD, Jirou Takezawa, MD, PhD, Naondo Sohara, MD, PhD, Satoru Kakizaki, MD, PhD, Hitoshi Takagi, MD, PhD, Masatomo Mori, MD, PhD

Gunma, Japan

Duodenal varices are relatively rare in patients with portal hypertension, but their rupture could be a serious and life-threatening event. We report an autopsy case of rupture of duodenal varices.

CASE REPORT

A 79-year-old woman had a history of liver cirrhosis with esophageal varices. In September 1997, endoscopic variceal ligation (EVL) and endoscopic injection sclerotherapy (EIS) were performed prophylactically with resultant shrinkage of the esophageal varices. In September 1998 tortuous duodenal varices were first diagnosed but were followed closely because of refusal of consent to any treatment. In September 2000 she underwent another EVL for ruptured esophageal varices. At 20 days after discharge, she was readmitted for hematemesis and melena. On admission, she was in a preshock state with severe anemia and was resuscitated with blood and fresh frozen plasma. Endoscopic examination identified bleeding from the varices in the second portion of the duodenum (Fig. 1). Hemostatic control was achieved by metallic clipping (Fig. 2). Several hours later, however, the duodenal varices ruptured again at a site close to the clipped area. Although EIS stopped the bleeding, the patient went into shock immediately after EIS and died. The postmortem confirmed the cause of death to be rupture of markedly dilated duodenal varices (Fig. 3).

DISCUSSION

Duodenal varices mainly are complications of intrahepatic portal hypertension, extrahepatic obstruction of portal vein, or splenic vein obstruction.[1]

Treatments for duodenal varices include endoscopic procedures (e.g., EVL, EIS, and clipping), surgery (e.g., variceal ligation, duodenal resection, and extrahepatic portosystemic shunts), and interventional radiologic procedures (IVR) (e.g., percutaneous transhepatic obliteration [PTO], transileocolic vein obliteration [TIO], transjugular intrahepatic portosystemic shunt [TIPS], and balloon-occluded retrograde transvenous obliteration [BRTO]).

According to a review of 16 reported cases,[2] placement of portosystemic shunts have been successful in the treatment of ruptured duodenal varices compared with surgical variceal ligation or duodenal resection.[2] The surgical shunt, however, carries approximately a 30% mortality rate.[2]

Because none of IVR has been investigated in large series of patients with duodenal varices, it is not clear which modality is more effective than the other modalities. PTO and TIO[3-5] or TIPS[6,7] are effective for a transient hemostasis of ruptured duodenal varices, but long-term outcome remains unclear. PTO or TIO often resulted in recanalization of obliterated veins or the appearance of new collaterals that feed esophageal or gastric varices.[8,9] Thus, PTO or TIO were preferably combined with other nonsurgical treatments, such as EIS or BRTO for esophageal or gastric varices to increase therapeutic effectiveness.[10,11] BRTO is less invasive and

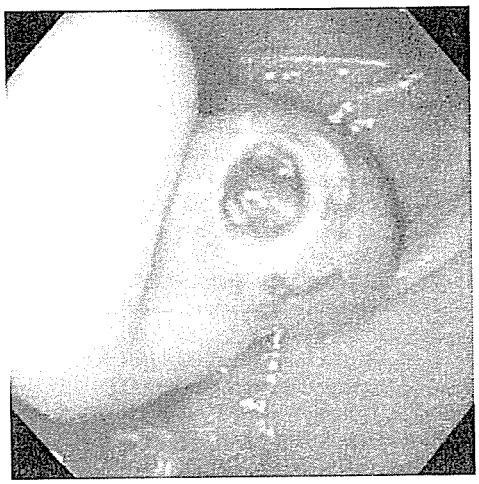


Figure 1. Endoscopic photograph showing varices in the second portion of the duodenum with hematocystic spotting.

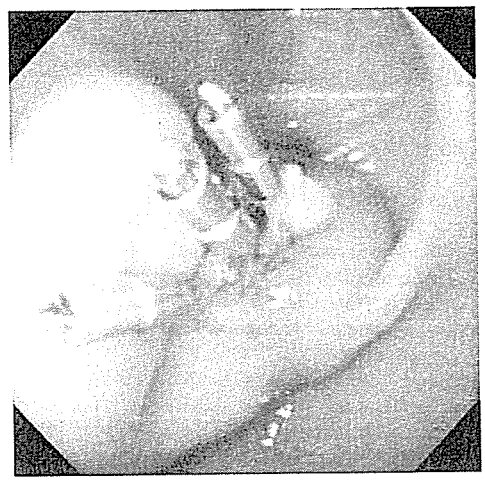


Figure 2. Endoscopic photograph showing varices in the second portion of the duodenum clamped with 3 metal clips.

more effective for gastric varices than TIPS.[12-15] The improvement rate of gastric fundal varices after TIPS is only 50%,[16] whereas BRTO can completely obliterate gastric varices in most cases.[12-15] In addition, TIPS has limitations, such as a significant mortality rate, shunt occlusion, and hepatic encephalopathy.[17] As for duodenal varices, BRTO or combined BRTO with TIO may be promising in the long term.[18-20] However, IVR demand considerable technical skill and are not always suitable for emergency cases.

Endoscopic procedures are less invasive compared with IVR, which are quicker and easier. Although successful obliteration of duodenal varices and resulting good long-term prognosis achieved by EIS[21] or EVL[22] was reported, neither EIS nor EVL showed consistent effectiveness.[23-32] EIS has several limitations, e.g., (1) EIS may induce leakage of sclerosing agents into the systemic circulation; (2) EIS carries a risk of penetration and perforation of the thin duodenal wall[28]; (3) duodenal hemostasis by balloon pressure is difficult to achieve anatomically[29]; and (4) duodenal varices, like gastric varices, represent a major portosystemic shunt and have more rapid and abundant blood flow.[30]

Endoscopic clipping and EVL achieve direct mechanical hemostasis and result in little local tissue injury and no systemic injury. Differing from EVL, the endoscopic-clip fixing device can be passed through the working channel and does not need removal of the endoscope for readying of the device. Moreover, EVL is challenging because it is difficult to achieve good visualization in the duodenum hampered by the presence of a banding chamber and to define limits between the varices and the duodenal papilla.[31] Identifying the papilla is critical to avoid its inclusion within banded tissue, because biliary obstruction can result from its banding.[32] However, there have been no comparative trials demonstrating that clipping had equal or superior efficacy to EVL or EIS for ruptured varices. Endoscopic clipping has been successfully applied to esophageal and fundal varices for hemostasis or prophylaxis with or without

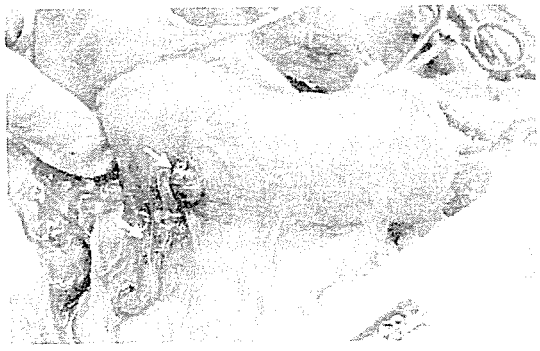


Figure 3. Autopsy findings. Note the presence of varices on the duodenal wall (*arrows*).

EIS[33-35] and duodenal varices, followed by BRTO[36] or laparoscopic resection.[37]

Because our patient was in poor physical condition, a safer and quicker treatment was considered to be preferable. Taken together, we chose endoscopic clipping as the first-line therapy for a transient hemostasis. After the patient's general status became better, we intended to try BRTO, after the example of successful treatment of BRTO after metal clipping for ruptured duodenal varices.[36]

Sauerbruch et al[38] reported a case of ruptured duodenal varices 7 weeks after sclerotherapy of esophageal varices. Their patient had thrombosis of the portocaval shunt and splenic vein, and underwent splenectomy. Our patient, however, had no thrombosis or past surgery. Although the details of the collateral veins are not available because of their collapse at post mortem, the 2 processes of development/rupture of duodenal varices in our case suggest post-EVL and/or EIS changes in portal blood flow as a possible mechanism. Occlusion of the esophageal varices and dilated draining veins may have worsened duodenal varices; representing another collateral route. Conversely, esophageal varices worsened after BRTO of duodenal varices.[19] Endoscopists should be aware that treatment of varices at

one location may result in the development or worsening of varices at other sites, and should carefully check for varices throughout the digestive tract.

REFERENCES

1. Amin R, Alexis R, Korzis J. Fatal ruptured duodenal varix: a case report and review of literature. Am J Gastroenterol 1985;80:13-8.
2. Khouqeer F, Morrow C, Jordan P. Duodenal varices as a cause of massive upper gastrointestinal bleeding. Surgery 1987;102:548-52.
3. Menu Y, Gayet B, Nahum H. Bleeding duodenal varices: diagnosis and treatment by percutaneous portography and transcatheter embolization. Gastrointest Radiol 1987;12:111-3.
4. Ohta T, Kitagawa T, Sohma M, et al. Duodenal varices treated successfully by percutaneous transhepatic obliteration: report of a case [in Japanese with English abstract]. Hokkaido Igaku Zasshi 1991;66:79-85.
5. Kakio T, Ito T, Sue K, et al. Hemostasis of gastric variceal hemorrhage by transileocoecal and transhepatic obliteration. Acta Med Okayama 1993;47:39-43.
6. Jonnalagadda SS, Quiason S, Smith OJ. Successful therapy of bleeding duodenal varices by TIPS after failure of sclerotherapy. Am J Gastroenterol 1998;93:272-4.
7. Vangeli M, Patch D, Terreni N, et al. Bleeding ectopic varices: treatment with transjugular intrahepatic porto-systemic shunt (TIPS) and embolisation. J Hepatol 2004;41:560-6.
8. Lunderquist A, Borjesson B, Owman T, et al. Isobutyl 2-cyanoacrylate (Bucrylate) in obliteration of gastric coronary vein and esophageal varices. AJR Am J Roentgenol 1978;130:1-6.
9. Smith-Laing G, Scott J, Long RG, et al. Role of percutaneous transhepatic obliteration of varices in the management of hemorrhage from gastroesophageal varices. Gastroenterology 1981;80:1031-6.
10. Tajiri T, Onda M, Yamashita K, et al. Interventional radiology for portal hypertension PTO. TIO [in Japanese with English abstract]. Nippon Geka Gakkai Zasshi 1996;97:70–7.
11. Hashizume M, Tsugawa K, Migo S, et al. Eradication of large gastric varices by sclerotherapy combined with percutaneous transhepatic obliteration. Hepatogastroenterology 1997;44:221-6.
12. Kanagawa H, Mima S, Kouyama H, et al. Treatment of gastric fundal varices by balloon-occluded retrograde transvenous obliteration. J Gastroenterol Hepatol 1996;111:51-8.
13. Hirota S, Matsumoto S, Tomita M, et al. Retrograde transvenous obliteration of gastric varices. Radiology 1999;211:349-56.
14. Chikamori F, Kuniyoshi N, Shibuya S, et al. Eight years of experience with transjugular retrograde obliteration for gastric varices with gastrorenal shunts. Surgery 2001;129:414-20.
15. Choi YH, Yoon CJ, Park JH, et al. Balloon-occluded retrograde transvenous obliteration for gastric variceal bleeding: its feasibility compared with transjugular intra hepatic Portosystemic shunt. Korean J Radiol 2003;4:109-16.
16. Sanyal AJ, Freedman AM, Luketic VA, et al. The natural history of portal hypertension after transjugular intrahepatic portosystemic shunts. Gastroenterology 1997;112:889-98.
17. Miller-Catchpole R. Diagnostic and therapeutic technology assessment. Transjugular intrahepatic portosystemic shunt (TIPS). JAMA 1995;273:1824-30.
18. Haruta I, Isobe Y, Ueno E, et al. Balloon-occluded retrograde transvenous obliteration (BRTO), a promising nonsurgical therapy for ectopic varices: a case report of successful treatment of duodenal varices by BRTO. Am J Gastroenterol 1996;91:2594-7.
19. Sonomura T, Horihata K, Yamahara K, et al. Ruptured duodenal varices successfully treated with balloon-occluded retrograde transvenous obliteration: usefulness of microcatheters. AJR Am J Roentgenol 2003;181:725-7.
20. Ota K, Okazaki M, Higashihara H, et al. Combination of transileocolic vein obliteration and balloon-occluded retrograde transvenous obliteration is effective for ruptured duodenal varices. J Gastroenterol 1999;34:694-9.
21. Yoshida Y, Imai Y, Nishikawa M, et al. Successful endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate following the recurrence of bleeding soon after endoscopic ligation for ruptured duodenal varices. Am J Gastroenterol 1997;92:1227-9.
22. Bosch A, Marsano L, Varilek GW. Successful obliteration of duodenal varices after endoscopic ligation. Dig Dis Sci 2003;48:1809-12.
23. Wang CS, Jeng LB, Chen MF. Duodenal variceal bleeding: successfully treated by mesocaval shunt after failure of sclerotherapy. Hepatogastroenterology 1995;42:59-61.
24. Ota K, Shirai Z, Masuzaki T, et al. Endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate for ruptured duodenal varices. J Gastroenterol 1998;33:550-5.
25. Hashiguchi M, Tsuji H, Shimono J, et al. Ruptured duodenal varices: an autopsy case report. Hepatogastroenterology 1999;46:1751-4.
26. Tazawa J, Sakai Y, Koizumi K, et al. Endoscopic ligation for ruptured duodenal varices. Am J Gastroenterol 1995;90:677-8.
27. Selcuk H, Boyvat F, Eren S, et al. Duodenal varices as an unusual cause of gastrointestinal bleeding due to portal hypertension: a case report. Turk J Gastroenterol 2004;15:104-7.
28. Barbish AW, Ehrinpreis MN. Successful endoscopic injection sclerotherapy of a bleeding duodenal varix. Am J Gastroenterol 1993;88:90-2.
29. Mikami K, Komatsu M, Ono T, et al. A case of rupture of duodenal varices due to idiopathic portal hypertension treated by endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate (Histoacryl) [in Japanese with English abstract]. Gastroenterol Endosc 1997;39: 949-53.
30. Wong RM, Ota S, Yazawa M, et al. Successful endoscopic variceal ligation in patient with bleeding from duodenal varices [in Japanese with English abstract]. Gastroenterol Endosc 1997;39:221-5.
31. Shiraishi M, Hiroyasu S, Higa T, et al. Successful management of ruptured duodenal varices by means of endoscopic variceal ligation: report of a case. Gastrointest Endosc 1999;49:255-7.
32. Silberzweig JE, Atillasoy EO, Sheiner PA, et al. Biliary obstruction caused by endoscopic band ligation of a duodenal varix. Am J Gastroenterol 1997;92:1060-2.
33. Miyoshi H, Shikata J, Tokura Y. Endoscopic clip of esophageal varices. Dig Endosc 1992;4:147-50.
34. Koutsomanis D. Endoscopic clipping for bleeding varices. Gastrointest Endosc 1994;40:126-7.
35. Hachisu T, Yamada H, Satoh S, et al. Endoscopic clip with a new rotatable clip-device and a long clip. Dig Endosc 1996;8:127-33.
36. Ohta M, Yasumori K, Saku M, et al. Successful treatment of bleeding duodenal varices by balloon-occluded retrograde transvenous obliteration: a transjugular venous approach. Surgery 1999;126:581-3.
37. Nakashima K, Sekiya R, Miyazaki T, et al. A case of duodenal varices: treatment by laparoscopic varicose vein resection [in Japanese with English abstract]. Nihon Naishikyo Geka Gakkai zasshi 2004;9:337-40.
38. Sauerbruch T, Weinzierl M, Dietrich HP, et al. Sclerotherapy of a bleeding duodenal varix. Endoscopy 1982;14:187-9.

Department of Internal Medicine, Kiryu Welfare General Hospital, Kiryu, Gunma, Japan (T.M.); Medicine and Molecular Science, Gunma University School of Medicine, Maebashi, Gunma, Japan (K.S.); Department of Internal Medicine, Kiryu Welfare General Hospital, Kiryu, Gunma, Japan (A.K., J.T.); Medicine and Molecular Science, Gunma University School of Medicine, Maebashi, Gunma, Japan (N.S., S.K., H.T., M.M.).

Reprint request: Ken Sato, MD, PhD, Department of Medicine and Molecular Science, Gunma University School of Medicine, 3-39-15, Showa, Maebashi, Gunma, 371-8511, Japan.

0016-5107/$32.00

doi:10.1016/j.gie.2005.08.031

LETTER TO THE EDITOR

The use of partial splenic artery embolization made it possible to administer interferon and ribavirin therapy in a liver transplant patient with fibrosing cholestatic hepatitis C complicated with thrombocytopenia

doi:10.1111/j.1432-2277.2006.00263.x

Hepatitis C virus (HCV) cirrhosis is a leading indication for orthotopic and living related liver transplantation (LT) [1]. However, the recurrence of HCV viremia is nearly universal after LT [2], and more than half of all patients develop clinical liver disease within a few years [3,4]. Although recurrent liver disease is clinically mild but progressive in most of these patients, a small number of these patients exhibit a fulminant course with progression to death, which is called fibrosing cholestatic hepatitis (FCH) [5,6]. Recently, interferon (IFN) and ribavirin have been shown to be effective in ameliorating hepatocellular injury in recurrent HCV after LT [7–9]. However, the adverse effects associated with this therapy, such as anemia, leukopenia, thrombocytopenia, make it necessary to discontinue this treatment interruption in a significant proportion of cases and the resulting poor adherence also reduces the efficacy of antiviral therapy in LT recipients in which a sustained virological response occurs in <20% of the treated patients [7–9]. It thus remains a big problem regarding how to manage these side effects in order to allow such patients to complete the IFN and ribavirin therapeutic regimen.

In this letter, we report a patient with FCH because of hepatitis C after LT, who successfully overcame thrombocytopenia as the result of IFN therapy after pretreatment with transcatheter partial splenic embolization (PSE). As a result, she was able to complete the IFN and ribavirin therapeutic regimen without any complications.

A 54-year-old woman with liver cirrhosis secondary to an HCV infection underwent a living related liver LT in August 2002. She was discharged from the hospital without any notable postoperative complications with immunosuppression consisting of FK506 (2 mg/day) and prednisolone (PSL) (5 mg/day). However, elevated liver function tests had been noted throughout the 1-year follow-up period after LT. In March 2003, at 7 months after LT, the first liver biopsy was performed. The liver specimen showed chronic active hepatitis caused by an HCV infection, mild activity and slight fibrosis with piecemeal necrosis. Then, IFN monotherapy with natural IFNα, three mega unit (MU) three times/week, was started. Two weeks later, however, her platelet count decreased to 30,000/mm^3, which thus made it necessary to discontinue the IFN therapy. Thereafter, the liver function tests remained abnormal. In August 2003, at 12 months after LT, her laboratory data showed markedly elevated levels of serum alkaline phosphatase (ALP) 1153 IU/l, r-glutanyl transpeptidase (rGTP) 2110 IU/l, and total bilirubin (T-Bil) 7.9 mg/dl, whereas the serum asparate aminotransferase (AST) 131 IU/l and the alanine aminotransferase (ALT) 75 IU/l level both moderately increased. On admission her laboratory data showed 2010 KIU/ml of HCV-RNA, genotype 2A, negative HBs-Ag and positive HBs-Ab. A liver biopsy performed on the following day revealed no evidence of rejection but progressive fibrosis with lobular regeneration, moderate inflammatory cell infiltration, and neo-ductular proliferation with hypercellularity. She was diagnosed to have FCH. Antiviral treatment, especially IFN and ribavirin combination should be administrated to such cases, as FCH is reported to be associated with a high viral load in the tissue and in the serum [6]. As a result, we decided to perform PSE prior to the therapy, as previous IFN treatment had been discontinued because of thrombocytopenia. A gel foam spongel with antibiotics (1 g of Cefazolin sodium) and contrast material was infused from the branch of splenic artery to embolize 50% of her spleen. No notable adverse effects were observed. As expected, her platelet count increased from 45 000 to 120 000/mm^3 2 weeks after PSE. We then started IFN and ribavirin combination therapy, IFN α2b, 3 MU three times/week and ribavirin 400 mg/day, in October 2003. As her platelet count did not deteriorate to <90 000/mm^3, we therefore completed the 8 month, combination therapy regimen in May 2004. The T-Bil, AST and ALT levels all normalized and the serum HCV-RNA also became negative 3 weeks after the initiation of the therapy. In May 2004, HCV RNA in the serum and liver tissue was still negative, and, as a result,

the treatment regimen was successfully completed. In May 2005, 12 months after the end of therapy, the patient's liver function tests were still normal and the serum HCV RNA level was continuously negative.

We herein showed our successful clinical observations in a patient with FCH because of hepatitis C after LT, who successfully overcame thrombocytopenia induced by IFN and ribavirin therapy by pretreatment with PSE, and thereafter was able to complete the IFN and ribavirin therapy for 8 months. This observation showed two important findings. Firstly, that IFN and ribavirin therapy was effective for FCH caused by hepatitis C. Secondly, PSE could be a promising optional treatment when thrombocytopenia causes IFN therapy to be discontinued.

Recently, IFN and ribavirin, when used as a therapy for post-LT hepatitis C, have been shown to reduce the serum ALT and HCV-RNA levels [10]. Especially, when a severe type of recurrent hepatitis C, such as FCH, occurred, these patients should be treated anti-viral therapy or a drastic reduction in immunosuppression, as the etiology is thought to be associated with high HCV viremia levels [4]. In our case, at 7 months after LT, we initially treated the patients with IFN monotherapy for her recurrent hepatitis C. However, the IFN therapy had to be discontinued within 2 weeks because of thrombocytopenia, despite the fact that both the transaminase levels and HCV viremia had been improving. Five months later, her next liver biopsy showed severe progressive fibrosis, a precirrhosis state, and FCH was thus diagnosed. Although several case reports have described that FCH improved after IFN and Ribavirin therapy, the effectiveness of this therapy for FCH remains unclear. However, since we did not have any other therapeutic option, we decided to treat her with IFN and ribavirin combination therapy. Fortunately, owing to the IFN and ribavirin therapy, her liver function test recovered to almost normal levels and HCV-RNA in the serum disappeared. The liver function data were observed to be continuously normal after the therapy for 12 months. In order to successfully complete the combination therapy, the thrombocytopenia had to improve. As, thrombopoietin preparation is not available at the present time, a splenectomy and PSE were thus considered as options to increase her platelet count. However, very few reports have demonstrated the effectiveness, safety and adverse effects of these therapies in post-LT patients. As splenectomy was reported to have such complications as a serious infection and sepsis with a high frequency, ranging from 0% to 60% of post-LT patients in previous reports [11–13]. On the other hand, PSE has been reported to be relatively safe in comparison to a splenectomy; only two serious adverse effects, one with abdominal pain due to PSE and one with hepatic artery thrombosis, out of 21 patients from five reports had been observed, and these complications were not fatal [14–18]. The usefulness of PSE before peg-interferon plus ribavirin has recently been reported in three LT patients [18]. In the other disease patients, PSE was usually safely performed, although some non fatal complications, such as fever, abdominal pain etc, were observed [19,20]. In addition, as the effect of PSE for thrombocytopenia was usually maintained for >6 months [21], we thus decided to perform PSE for the pretreatment of IFN and ribavirin therapy. As expected, the platelet count increased, and we could complete the 8-month regimen for IFN and ribavirin therapy. In conclusion, IFN and ribavirin therapy was effective for FCH because of hepatitis C, and PSE is therefore considered to be a promising and effective adjuvant modality, in LT patients with hepatitis C recurrence, which allows them to overcome thrombocytopenia before undergoing IFN and ribavirin therapy.

Naondo Sohara, Hitoshi Takagi, Satoru Kakizaki, Ken Sato and Masatomo Mori
Department of Medicine and Molecular Science, Gunma University Graduate School of Medicine, 3-39-15 Showamachi, Maebashi, Gunma 371-8511, Japan

References

1. Berengure M, Crippin J, Gish R, *et al.* A model to predict severe HCV-related disease following liver transplantation. *Hepatology* 2003; **38**: 34.
2. Ascher NL, Lake JR, Emond J, Roberts J. Liver transplantation for hepatitis C virus-related cirrhosis. *Hepatology* 1994; **20**: 24S.
3. Gane EJ, Portmann BC, Naoumov NV, *et al.* Long-term outcome of hepatitis C infection after liver transplantation. *N Engl J Med* 1996; **334**: 815.
4. Schluger LK, Sheiner PA, Thung SN, *et al.* Severe recurrent cholestatic hepatitis C following orthotopic liver transplantation. *Hepatology* 1996; **23**: 971.
5. Zylberberg H, Carnot F, Mamzer MF, Blancho G, Legendre C, Pol S. Hepatitis C virus-related fibrosing cholestatic hepatitis after renal transplantation. *Transplantation* 1997; **63**: 158.
6. Dickson RC, Caldwell SH, Ishitani MB, *et al.* Clinical and histological patterns of early graft failure due to recurrent hepatitis C in four patients after liver transplantation. *Transplantation* 1996; **61**: 701.
7. Samuel D, Bizollon T, Feray C, *et al.* Interferon-alpha 2b plus ribavirin in patients with chronic hepatitis C after liver transplantation: a randomized study. *Gastroenterology* 2003; **124**: 642.
8. Shakil AO, McGuire B, Crippin J, *et al.* A pilot study of interferon alfa and ribavirin combination in liver

transplant recipients with recurrent hepatitis C. *Hepatology* 2002; **36**: 1253.
9. Roche B and Samuel D. Treatment of hepatitis B and C after liver transplantation. Part 2, hepatitis C. *Transpl Int* 2005; **17**: 759.
10. Bizollon T, Palazzo U, Ducerf C, *et al.* Pilot study of the combination of interferon alfa and ribavirin as therapy of recurrent hepatitis C after liver transplantation. *Hepatology* 1997; **26**: 500.
11. Shimada M, Ijichi H, Yonemura Y, *et al.* The impact of splenectomy or splenic artery ligation on the outcome of a living donor adult liver transplantation using a left lobe graft. *Hepatogastroenterology* 2004; **51**: 625.
12. Cescon M, Sugawara Y, Takayama T, *et al.* Role of splenectomy in living-donor liver transplantation for adults. *Hepatogastroenterology* 2002; **49**: 721.
13. Troisi R, Hesse UJ, Decruyenaere J, *et al.* Functional, life-threatening disorders and splenectomy following liver transplantation. *Clin Transplant* 1999; **13**: 380.
14. Sockrider CS, Boykin KN, Green J, *et al.* Partial splenic embolization for hypersplenism before and after liver transplantation. *Clin Transplant* 2002; **16**(Suppl. 7): 59.
15. Uflacker R, Selby JB, Chavin K, Rogers J, Baliga P. Transcatheter splenic artery occlusion for treatment of splenic artery steal syndrome after orthotopic liver transplantation. *Cardiovasc Intervent Radiol* 2002; **25**: 300.
16. Herrero JI, Sangro B, Quiroga J, *et al.* Partial splenic embolization in the treatment of thrombocytopenia after liver transplantation. *Transplantation* 1997; **63**: 482.
17. Firat A, Boyvat F, Moray G, Aytekin C, Karakayali H, Haberal M. Comparison of two different percutaneous splenic artery interventions in the treatment of hypersplenism: preliminary report. *Transplant Proc* 2005; **37**: 1094.
18. Gil-Grande R, Moreno J, Foruny JR, *et al.* Partial splenic embolization for the treatment of hypersplenism in liver transplated patients with hepatitis C virus recurrence before peg-interferon plus ribavirin. *Transplantation* 2005; **79**: 1634.
19. Palsson B, Hallen M, Forsberg AM, Alwmark A. Partial splenic embolization: long-term outcome. *Langenbecks Arch Surg* 2003; **387**: 421.
20. Mozes MF, Spigos DG, Pollak R, *et al.* Partial splenic embolization, an alternative to splenectomy – results of a prospective, randomized study. *Surgery* 1984; **96**: 694.
21. Romano M, Giojelli A, Capuano G, Pomponi D, Salvatore M. Partial splenic embolization in patients with idiopathic portal hypertension. *Eur J Radiol* 2004; **49**: 268.